

Physiology of ureter and renal pelvis

Transport of urine from renal pelvis to bladder *active* process requiring:

- Pacemaker cells
- Propagation of electrical impulse
- Adequate mucosal coaption
- Intraluminal pressure > bladder pressure

Electrical activity

Development of resting membrane potential dependent on K⁺

Inside K⁺; outside Na⁺: K⁺ leaks out of cell down concentration gradient through open potassium channels.

Electrochemical gradient thus set up with **inside negative** cf. outside, stopping further K⁺ efflux

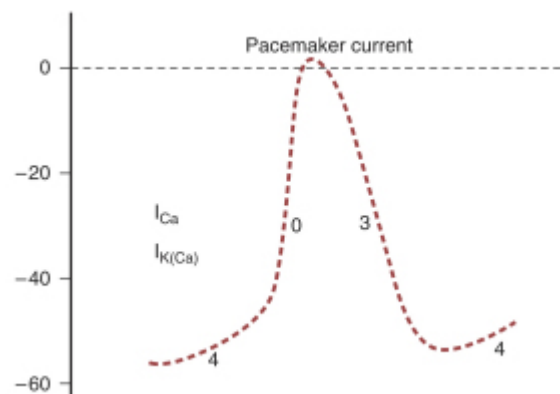
Typical resting membrane potential of ureteral cell = -30mV to -70mV

Depolarisation in pacemaker cells:

Pacemaker cells located near pelvicalyceal border

Pacemaker cells differ from ordinary cells in that **Na⁺ permeability is unchanged by depolarisation**

Opening and slow closure of Ca²⁺ channels offset by opening and slow closure of K⁺ channels alone, leading to rhythmic depolarisation and repolarisation



Believed that prostaglandins and tachykinins (prostanoids) regulate/amplify pacemaker cell activity

Propagation of depolarisation via intermediate/gap junctions; speed 2-6cm/s [NB. ureter 22-30cm long in adults]; frequency of driven action potentials 3-5/min

Depolarisation in non-pacemaker cells:

Opening of Na⁺ and Ca²⁺ channels; closure of K⁺ channels

Na⁺ in fast, Ca²⁺ in slow – rapid depolarisation then plateau

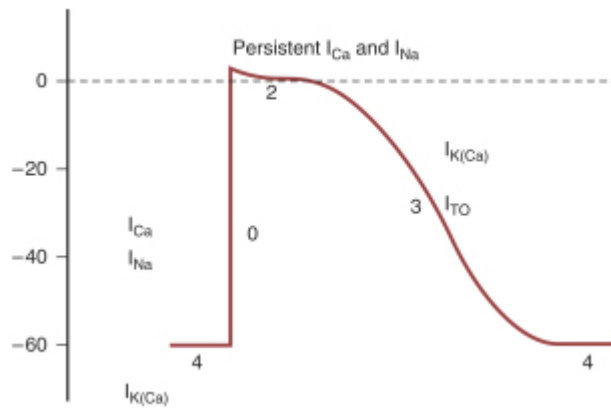
Raised cytosolic Ca²⁺ stimulates smooth muscle contraction*

Raised intracellular Ca²⁺ re-opens voltage-gated K⁺ channels

Rapid K⁺ efflux repolarises cell**

* depolarisation also transmitted via tubules to endoplasmic reticulum stimulating Ca release

** restoration of electrolyte balance by Na⁺/K⁺ ATPase and cAMP (drives Ca²⁺ back into endoplasmic reticulum)



Mediators of peristaltic activity

	Contraction	Relaxation
Neuromodulators	Alpha-adrenoceptor Tachykinins Histamine Angiotensin ? cholinergic stimulation ? serotonergic PGF2a	Calcitonin gene-related peptide Beta-adrenoceptor PGE1 and PGE2*
Drugs	? opiates	Progesterone Calcium channel blockers Potassium channel openers (nicorandil)

* Surprisingly NSAIDs have not been shown to affect frequency or strength of ureteric contraction. They are believed to work by inhibiting prostaglandin mediated afferent arteriolar dilatation (reducing pelvic pressure and therefore pain) and reducing ureteric inflammation

Urine transport

At normal urinary flow boluses of urine are formed by peristaltic waves

Resting ureteric pressure = 0 - 5 cm water

Peristaltic waves = 20 – 80 cm water

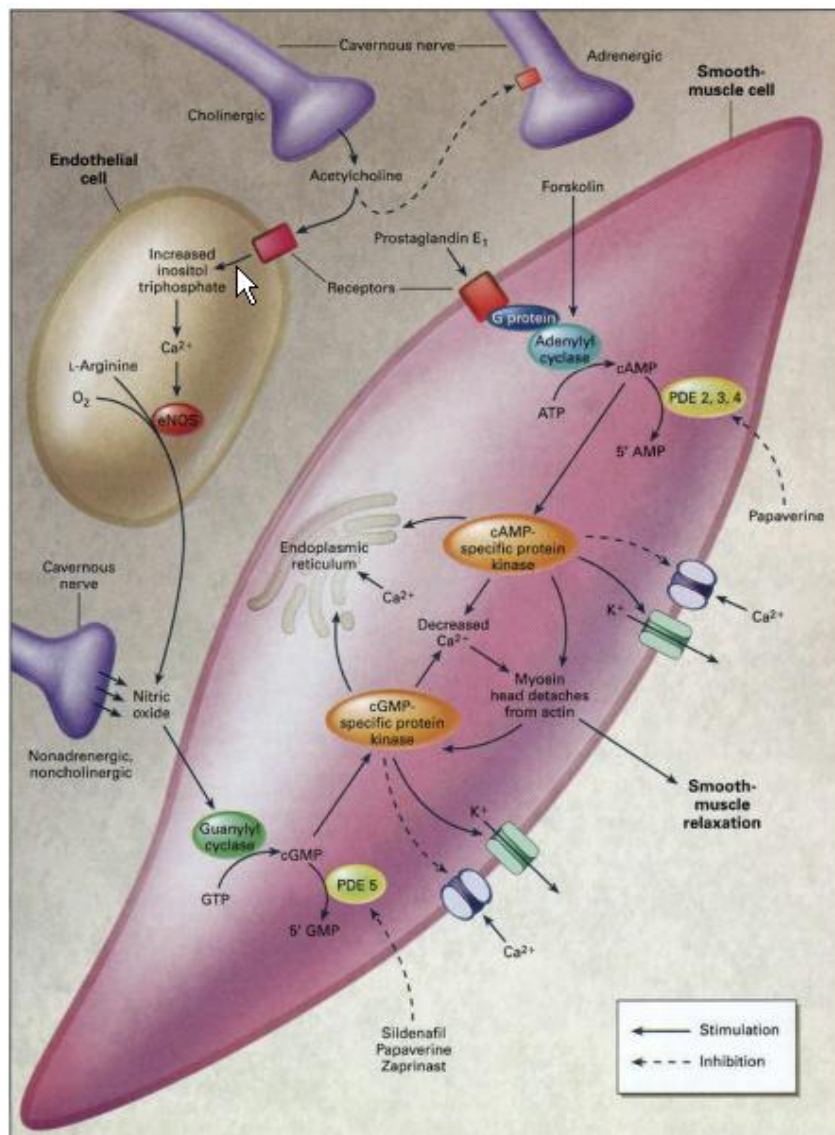
Frequency = 2 – 6 times per minute

At very high flow rates, mucosal walls do not coapt and urine transported as a continuous column

Decompensation when bladder pressure > 40cm water

Appendix

Smooth muscle relaxation



Smooth muscle contraction

